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Environmental, Dietary, and Behavioral Factors Distinguish Chinese Adults with High Waist-to-Height Ratio with and without Inflammation[1–3]

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Abstract

Background: The environmental and behavioral risk factors associated with central obesity and/or inflammation in populations exposed to both obesogenic and pathogenic environments remain unclear.

Objectives: We tested which of the characteristics distinguished 3 risk groups—high waist-to-height ratio (WHtR; >0.5) without inflammation [high-sensitivity C-reactive protein (hs-CRP) <3 mg/L], normal WHtR (≤0.5) with inflammation (hs-CRP: 3–10 mg/L), and high WHtR with inflammation—from the referent group with normal WHtR without inflammation and, secondarily, which factors differed between the groups with high WHtR with and without inflammation.

Methods: The analytic sample included 8068 adults participating in the China Health and Nutrition Survey in 2009. Adjusted multinomial and logistic regression models were used to assess the risk of being in one of the ''unhealthy'' groups compared with the referent group.

Results: Men with high WHtR with and without inflammation were more likely to live at higher urbanicity (57–63%) and have higher incomes (26–42%) and household sanitation (26–67%) and were >40% less likely to have high physical activity than the healthy referent group. Men with high WHtR with inflammation had higher odds of infectious symptoms than those with high WHtR without inflammation (OR: 1.73; 95% CI: 1.15, 2.61). Women with high WHtR without inflammation were less likely to have high household sanitation (44%) or perform high levels of physical activity (24%) and were 34% more likely to consume more fiber than the healthy referent group. Women with high WHtR and inflammation were more likely than those with high WHtR without inflammation to have infectious symptoms (OR: 1.45; 95% CI: 1.01, 2.07) and less likely to have higher fiber intake (OR: 0.77; 95% CI: 0.60,1.00) or physical activity (OR: 0.55; 95% CI: 0.41, 0.73).

Conclusion: These results document different underlying pathogenic and obesogenic risk factors for visceral adiposity with and without inflammation in Chinese adults, suggesting that context-specific approaches may be needed to prevent and treat inflammation. *J Nutr* 2015;145:1335–44.

Keywords: inflammation, waist-to-height ratio, China, central obesity, C-reactive protein

Introduction

Low-grade inflammation has been associated with the development of a number of chronic diseases, including cardiovascular disease and type 2 diabetes (1–3). Although the exact mechanisms linking inflammation and cardiometabolic disease have yet to be elucidated, overweight and obesity have been implicated as important mediating factors. Overweight and obesity are strongly associated with increased odds of having elevated C-reactive protein (CRP)[8], a commonly measured marker of systemic inflammation (4, 5). More recently, waist-to-height

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[3] Supplemental Tables 1 and 2 are available from the ''Online Supporting Material'' link in the online posting of the article and from the same link in the online table of contents at http://jn.nutrition.org.

[8] Abbreviations used: CHNS, China Health and Nutrition Survey; CRP, C-reactive protein; hs-CRP, high-sensitivity C-reactive protein; ID, infectious disease; WHtR, waist-to-height ratio.

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ratio (WHtR), a marker of central adiposity, was shown to be more strongly associated with CRP than BMI or waist circumference alone (6, 7), particularly in younger individuals and those with lower BMI (8). WHtR may better reflect visceral adipose tissue accumulation and thus the production of proinflammatory cytokines, such as IL-6 and TNF-α, which stimulate CRP. Indeed, several studies suggested that differences in visceral adiposity and resulting inflammatory profiles may distinguish "metabolically healthy obese" individuals from those considered "metabolically unhealthy" (9–11).

Differential exposure to behavioral and environmental factors is thought to underlie these metabolically healthy vs. unhealthy obesity phenotypes and the transition from one subtype to another (10, 12). However, much of this research has focused on the United States and other Western countries (13). Relatively less is known about the factors associated with visceral adiposity and inflammatory profiles in populations characterized by differing obesity phenotypes (13) and simultaneous exposure to obesogenic and pathogenic factors (14–16). Previous research in developing countries, such as China and the Philippines, that have seen rapid transitions in environment, diet, and disease burden has shown that a substantial proportion of the population may have moderately elevated CRP even in the absence of visceral adiposity (16) or, conversely, may have low CRP concentrations even at levels of visceral adiposity similar to those associated with elevated CRP in US samples (14). Such results suggest that the relation between visceral adiposity and inflammation may differ in developing country contexts and that those with high visceral adiposity or inflammation may represent distinct groups with differing environmental exposures.

To explore how differing environments may contribute to central obesity and/or inflammation in a developing country context, we examined the environmental and behavioral factors that distinguish 3 risk groups (high WHtR without inflammation, high WHtR with inflammation, and normal WHtR with inflammation) from a healthy referent group (normal WHtR and no inflammation) among Chinese men and women aged 18–99 y participating in the China Health and Nutrition Survey (CHNS). We tested whether demographic factors, environmental exposures, and health behaviors differed between these groups, with the hypotheses that *1*) those with higher pathogenic exposures will be at greater risk of inflammation in the absence of high WHtR, *2*) those who consume a more Western diet and with obesogenic exposures will be at greater risk of high WHtR with or without inflammation, and *3*) those with both pathogenic and obesogenic exposures will be at greatest risk of high WHtR with inflammation. Finally, because those with both high WHtR and inflammation may be considered the group most at risk of cardiometabolic morbidity and mortality (17), we examined what distinguishes this group from those with high WHtR but normal high-sensitivity CRP (hs-CRP) concentrations.

Methods

Sample

The data used in this study come from the 2009 wave of the CHNS. As previously described in detail (18), the CHNS is a longitudinal study of environment, health, and nutrition with measures at the community, household, and individual levels with 8 waves of data collection from 1989 to 2009. Participants include 14,000 individuals from >4400 households across 9 diverse provinces (Guangxi, Guizhou, Heilongjiang, Henan, Hubei, Hunan, Jiangsu, Liaoning, and Shandong). The original 1989 survey used a multistage random-cluster design to select a stratified probability sample that was diverse in urban/rural location and socioeconomic status. Participating communities were selected from 2 cities, 1 large and 1 small, and 4 counties, stratified by income, in each province. Within cities, 2 urban and 2 suburban communities were randomly selected; within counties, 1 community in the capital city and 3 rural villages were randomly chosen. Twenty households per community or rural village were then randomly selected for participation.

Data were collected in the participating households and included sociodemographic surveys, anthropometric measurements, 3 d of 24-h dietary recall and weighed food inventory collection, and household environmental measures. In the 2009 wave, blood samples were collected in participants over the age of 7 y at neighborhood clinics or, in cases in which participants were unable to attend the clinic visit, at home. The analytic sample includes 8068 adults (3770 men and 4298 women), aged 18–99 y, with measured hs-CRP and waist circumference. Subjects were excluded if they were pregnant (n = 58) or had hs-CRP >10 mg/L (n = 309), a commonly used indicator of current infection (19). This study protocol and analysis were approved by the institutional review board at the University of North Carolina at Chapel Hill, the China-Japan Friendship Hospital, Ministry of Health, and the Institute of Nutrition and Food Safety, China Centers for Disease Control. Subjects gave informed consent for participation.

Measures

hs-CRP. Fasting blood samples were collected from participants by venipuncture. Serum hs-CRP concentrations were measured using a high-sensitivity immunoturbidmetric method (Hitachi 7600 automated analyzer) with Denka Seiken reagents by a nationally accredited medical laboratory in Beijing (International Organization for Standardization 15189:2007). This assay has a range of 0.1–320 mg/L and a CV <7.0% across assays. The distribution of hs-CRP was highly skewed even after logarithmic transformation; consequently, hs-CRP was dichotomized into 2 levels representing normal (<3 mg/L) and moderate (3–10 mg/L) elevation (19).

WHtR. Two trained health workers collected height and waist circumference following standard protocols. Height was measured without shoes to the nearest 0.2 cm using a portable stadiometer and averaged across all repeated measures for analyses. Waist circumference was measured midway between the lowest rib and the iliac crest using a nonelastic tape. WHtR was created by dividing waist circumference (in cm) by height (in m). High WHtR was defined as a ratio >0.5 for both men and women (20). Participants were categorized into 4 groups defined by their dichotomous inflammation and WHtR status: healthy (WHtR ≤0.5 and hs-CRP <3 mg/L), high WHtR without inflammation (WHtR >0.5 and hs-CRP <3 mg/L), inflammation without high WHtR (WHtR ≤0.5 and hs-CRP of 3–10 mg/L), and high WHtR with inflammation (WHtR >0.5 and hs-CRP of 3–10 mg/L).

Sociodemographic, environmental, and behavioral factors. We examined whether the groups differed in urbanicity, income, and age to describe the regional and demographic risk factors for high WHtR and/or inflammation. Urbanicity was defined by using a multidimensional 12-component urbanization index capturing community-level physical, social, cultural, and economic environments (21). Scores from the scale ranged from 30.4 to 106.6, and tertiles representing low, middle, and high urbanicity were used in analysis. Income was derived from total household income, measured in yuan and similarly divided into tertiles representing low, medium, and high incomes. Region, north vs. south, was included because of documented differences in health indicators and longevity by region in China (22). Age was categorized into ∼10-y periods (18–29.9, 30–39.9, 40–49.9, 50–59.9, 60–69.9, and ≥70 y) to account for potential nonlinearity in the relation between age and WHtR.

A number of behavioral and environmental factors previously associated with inflammation and/or visceral adiposity were examined including the following: fat and fiber intake, physical activity, alcohol consumption, smoking, household sanitation, and infectious illness symptoms. Daily fat and fiber intakes were used as a marker of a westernized diet. Fat and energy were derived from three 24-h recalls taken on consecutive days and estimated from the 2002/2004 Chinese food-composition table (23). Diets were considered high in fat if ≥30% of the total daily energy intake was derived from fat (24). Fiber intake was estimated from USDA values and adjusted for energy intake. A categorical variable was defined on the basis of the sample distribution of fiber in grams per 100 kcal into <25th percentile, 25th–75th percentile, and >75th percentile, representing intakes of <0.75, 0.75–1.32, or >1.32 g/100 kcal, respectively. Physical activity was reported with the use of a detailed 7-d physical activity recall instrument. Activities were assigned metabolic equivalent values by using the *Compendium of Physical Activities* (25), which was previously validated for use in China (26). Metabolic equivalent hours per week of total physical activity were calculated from the sum of domestic, occupational, active leisure, and travel physical activity and categorized into quartiles for analysis. Alcohol consumption was separately defined for men and women because of pronounced sex differences in the frequency and amount of alcohol consumption. For men, alcohol consumption was coded as a 3-level variable (none, low, and high) representing nondrinker, ≤7 drinks/wk, and >7 drinks/wk, respectively. For women, alcohol consumption was coded as nondrinkers vs. any consumption. Servings were defined as 1 bottle of beer or 50 g wine or liquor. Smoking was included as a dichotomous variable based on whether participants were currently smoking at the 2009 survey.

Following previous research (14), we created a household sanitation index to measure potential exposure to environmental pathogens. The index included 7 variables previously linked to inflammation in this sample (16): availability of water (i.e., in-home tap, in-yard tap, in-yard well, or other), water source (i.e., ground, open well, river/lake, or water plant), location of toilet (i.e., in home, public, open pit, none, or other), amount of excrement visible around the home, type of fuel used as the primary cooking fuel (i.e., particulate-generating vs. non–particulate-generating), presence of livestock, and median density of cars within the community. Variables measuring presence or absence (livestock exposure, particulate-generating cooking fuel, and median density of cars) were scored on a 2-point scale, whereas the remaining variables were scored on a 4-point scale. The resulting scale had a possible range of 7–22, with higher values for each variable representing better household sanitation, and a Cronbach's α of 0.70, reflecting a relatively high degree of reliability (27). Because of the skewed distribution of index scores and the previously documented nonlinear association between household measures and hs-CRP (16), tertiles of the index were used in analysis to represent low, moderate, and high household sanitation. Participants' self-reported infectious illness symptoms in the 24 h preceding blood collection were also included as a measure of pathogen burden. At the time of blood collection, participants were asked whether they had experienced any illness in the past 24 h and about the occurrence of 7 sets of symptoms/conditions (fever/sore throat/cough/asthma, diarrhea/ stomachache, headache/dizziness, rash/dermatitis, heart disease/chest pain, other infectious diseases (IDs), and other noncommunicable disease). This variable was dichotomized into any symptoms vs. no symptoms and was assigned a positive value if participants had experienced fever/cough/sore throat, diarrhea, or other infectious illness in the preceding 24 h.

Statistical analysis

We first examined age patterns in high WHtR with and without inflammation and moderate inflammation with and without high WHtR for each sex using a Pearson chi-square test of trend. We then examined pairwise differences in demographic, behavioral, and environmental variables separately for men and women using *t* tests for continuous outcomes and chi-square tests for categorical outcomes. These are presented for the 2 referent groups of interest, the healthy group and those with high WHtR without inflammation. We used multinomial logistic regression models to compare the factors independently, placing individuals at greater or lesser risk of being in 1 of the 3 "unhealthy" groups compared with the healthy referent group. We conducted separate models for the sociodemographic variables and the behavioral and environmental variables to distinguish between the more distal measures that may reflect geographical differences and/or unmeasured lifestyle and environmental variables and the more proximal individual measures of health behaviors and environmental exposures. We conducted similarly specified logistic regression models to explicitly compare the factors distinguishing those with both high WHtR and inflammation from the referent group of high WHtR without inflammation. Finally, we conducted a sensitivity analysis simultaneously including all sociodemographic and behavioral variables in our multinomial and logistic models to assess whether the results would change substantially. All models controlled for age. All analyses were conducted by using STATA version 13 (StataCorp). Significance was considered to be $P < 0.05$.

Results

Sample characteristics. The prevalences of both moderate inflammation (hs-CRP: 3–10 mg/L) and high WHtR (WHtR >0.5) increased with age in this sample of Chinese men and women (**Figure 1**; *P*-trend < 0.001 for all tests). The proportion of the sample with high WHtR and inflammation increased with age in both men and women (*P*-trend < 0.001). In contrast, the proportion of the sample with normal WHtR and inflammation (WHtR ≤0.5, hs-CRP of 3–10 mg/L) increased significantly with age in men (*P*-trend = 0.008) but not women (*P*-trend = 0.44). Nearly all sociodemographic and behavioral characteristics differed significantly between the healthy referent group and the inflammation and/or obesity groups and between groups with high WHtR without inflammation, inflammation with normal WHtR, and inflammation with high WHtR in bivariate analysis (**Table 1**).

Risk factors for high WHtR and/or inflammation compared with the healthy referent group. In multivariable models, we examined whether the 3 risk groups could be distinguished from the healthy referent group on the basis of sociodemographic and geographic factors (**Table 2**, model 1) and behavioral factors (Table 2, model 2). Men with high WHtR with and without inflammation shared similar sociodemographic characteristics; both groups were more likely to live at middle and high levels of urbanicity and to have higher levels of income compared with the healthy referent group. Behavioral risk factors differed between men with high WHtR with and without inflammation. Men with high WHtR without inflammation were significantly more likely to have moderate and high levels of alcohol consumption and less likely to perform high levels of physical activity or to smoke in comparison to the healthy referent group. Men with both high WHtR and inflammation were more likely to have ID symptoms, more likely to have better household sanitation, and less likely to have high levels of physical activity than the referent group. Men with inflammation but normal WHtR were only distinguished from the healthy referent group by being significantly more likely to have had ID symptoms in the 24 h preceding blood collection.

Both demographic and behavioral variables differed between women with high WHtR with and without inflammation when compared with the healthy referent group. Women with high WHtR without inflammation were less likely to live in highly urban areas, have high household sanitation, and have moderate and high levels of physical activity and were more likely to consume higher amounts of fiber than the referent group. Women with both high WHtR and inflammation were more likely to live at middle levels of urbanicity and were less likely to perform any level of physical activity compared with the healthy referent group. As with men, women with inflammation but normal WHtR were more likely to have experienced ID symptoms.

What risk factors distinguish the groups with high WHtR with and without inflammation? Finally, to test the risk factors distinguishing those with high WHtR and inflammation from those with high WHtR without inflammation, we conducted logistic regression models explicitly comparing these groups

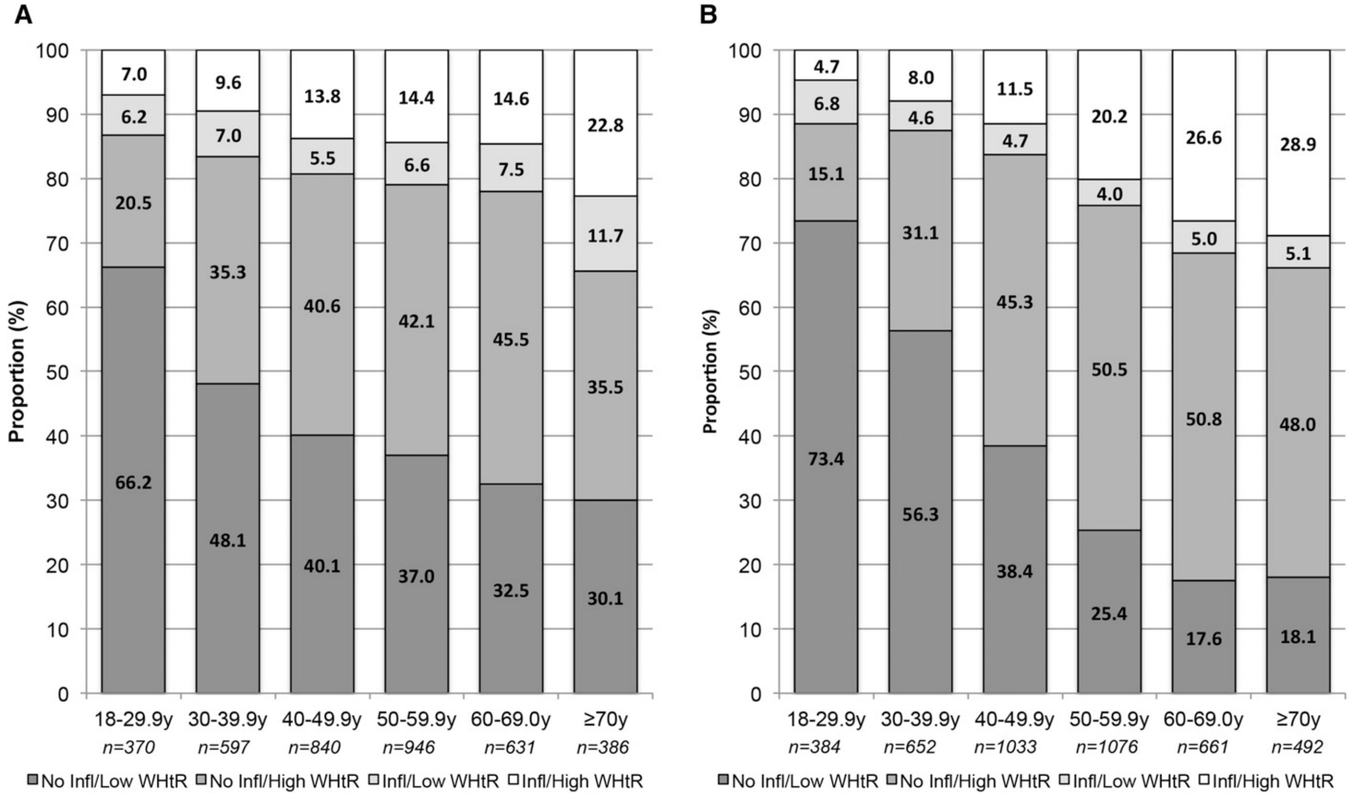

FIGURE 1 Prevalence of moderate inflammation and high WHtR in Chinese men (A) and women (B). Numbers within the bars represent age group percentages in each risk group. hs-CRP, high-sensitivity C-reactive protein; Infl, inflammation; Infl/High WHtR, hs-CRP of 3–10 mg/L and WHtR >0.5; Infl/Low WHtR, hs-CRP of 3–10 mg/L and WHtR ≤0.5; No Infl/High WHtR, WHtR >0.5 and hs-CRP <3 mg/L; No Infl/Low WHtR, hs-CRP <3 mg/L and WHtR ≤0.5; WHtR, waist-to-height ratio.

(**Table 3**). No distal factors differed between men in these groups (model 1) and only ID symptoms in the past 24 h differed between the groups, with men with both high WHtR and inflammation more likely to have had ID symptoms (model 2). In contrast, several distal and proximal characteristics distinguished women in the inflammation with high WHtR groups from the high WHtR without inflammation referent group. Compared with those with high WHtR without inflammation, women with high WHtR and inflammation had greater odds of living at high urbanicity and having ID symptoms in the past 24 h and lower odds of consuming the highest amounts of fiber or having the highest levels of physical activity. Interestingly, age did not differ significantly between these 2 groups in men or women.

Sensitivity analysis. The results obtained from the sensitivity analysis comparing full models that included all of the sociodemographic and environmental/behavioral variables simultaneously were generally consistent with those found with the separate multinomial and logistic models (**Supplemental Tables 1** and **2**, respectively). A few results, such as the effect of urbanicity in distinguishing group membership, were somewhat attenuated in the full multinomial and logistic models. These effects suggested that the patterning of several exposures, such as fiber intake or pathogen burden, differ by urban/rural location and/or income.

Discussion

We identified a number of sociodemographic, environmental, and behavioral risk factors that differed across the inflammation and high WHtR risk groups in Chinese men and women. As we hypothesized, pathogen burden, as indicated by ID symptoms, distinguished men and women with inflammation but normal WHtR from the healthy referent group. Obesogenic exposures were associated with a greater likelihood of being in one of the high WHtR groups (with and without inflammation) for both men and women. Men with high WHtR with and without inflammation were both more likely to live at higher urbanicity and to have higher incomes and less physical activity than the healthy referent group. Similarly, women with high WHtR with and without inflammation were more likely to perform less physical activity than the healthy referent group. Exposure to both obesogenic and pathogenic factors was associated with a greater likelihood of being in the group with high WHtR with inflammation for men and women. Men and women with high WHtR and inflammation were more likely to have infectious illness symptoms in the past 24 h in addition to the obesogenic exposures of living at higher urbanicity or performing less physical activity. Women with high WHtR and inflammation, for example, were more likely than those with high WHtR without inflammation to live at higher urbanicity and to have infectious symptoms, lower fiber intake, and lower physical activity. To our knowledge, our study is the first to examine the association between a wide range of obesogenic and pathogenic risk factors and WHtR in Chinese adults and to distinguish between those with high WHtR alone or with concurrent inflammation. Our results suggest that these groups may reflect distinct segments of the population with unique patterns of exposure and highlight the importance of examining the dynamics of both central obesity and inflammation in societies undergoing rapid changes in diet, activity, and disease burdens.

Previous studies have shown that Asian populations tend to have higher visceral adiposity and greater metabolic consequences

TABLE 1 Characteristics of Chinese men and women participating in the CHNS by WHtR and inflammation group[1]

	Men				Women			
	No infl/norm WHtR (n = 1540)	No infl/high WHtR (n = 1450)	Infl/norm WHtR (n = 265)	Infl/high WHtR (n = 515)	No infl/norm WHtR (n = 1524)	No infl/high WHtR (n = 1844)	Infl/norm WHtR (n = 206)	Infl/high WHtR (n = 724)
Age, y	47.1 ± 15.5	52.3 ± 13.8*	53.0 ± 16.0*	54.3 ± 14.2*†	53.8 ± 13.1	49.5 ± 15.9*	57.9 ± 13.1*†	53.6 ± 17.1*†
hs-CRP, mg/L	0.79 ± 0.71	1.06 ± 0.7*	5.03 ± 2.1*†	4.67 ± 1.89*†	0.62 ± 0.69	0.97 ± 0.70*	4.50 ± 1.76*†	4.81 ± 1.95*†
Waist circumference, cm	76.3 ± 6.0	91.4 ± 7.0*	76.4 ± 6.0†	93.3 ± 7.4*†	71.8 ± 5.3	86.5 ± 7.2*	72.5 ± 5.5†	89.6 ± 8.4*†
Height, cm	168 ± 6.9	167 ± 6.7*	167 ± 6.8†	167 ± 6.3	157 ± 6.0	155 ± 6.4*	157 ± 6.4†	154 ± 6.2*†
BMI, kg/m^2	21.2 ± 2.3	25.2 ± 2.9*	21.1 ± 2.5†	25.8 ± 3.2*†	20.9 ± 2.2	24.6 ± 2.9*	21.2 ± 2.5†	26.1 ± 3.6*†
Income, ×1000 yuan	37.4 ± 45.5	40.8 ± 47.2	32.1 ± 68.7†	44.7 ± 62.0*	39.2 ± 41.7	36.0 ± 45.2	39.8 ± 44.8	35.8 ± 41.4
Urbanicity,[2] % (n)								
Middle	30.7 (472)	34.3 (498)*	34.3 (91)	34.0 (175)*	30.8 (469)	35.1 (648)	30.6 (63)	34.4 (249)*
High	31.4 (483)	36.4 (528)*	29.8 (79)†	38.5 (198)*	37.8 (575)	30.9 (569)*	40.3 (83)†	38.0 (275)†
North resident,[3] % (n)	37.7 (581)	47.5 (689)*	39.3 (227)†	44.0 (227)*	40.2 (612)	45.9 (846)*	40.3 (83)	40.5 (293)†
Sanitation index score[4]	17.6 ± 3.6	18.3 ± 3.5*	17.4 ± 3.6†	18.7 ± 3.9*	18.3 ± 3.5	17.8 ± 3.6*	18.3 ± 3.5	18.5 ± 3.4†
ID symptoms,[5] % (n)	4.5 (69)	4.7 (68)*	12.1 (32)*†	7.8 (40)*†	5.7 (86)	5.2 (96)	10.3 (21)*†	7.6 (55)†
High fat intake,[6] % (n)	47.6 (724)	52.4 (750)*	51.2 (133)	54.6 (276)*	55.7 (831)	52.8 (959)	60.0 (120)	59.6 (423)†
Fiber intake, g/d	24.5 ± 13.7	25.6 ± 14.4	23.4 ± 14.4	23.7 ± 12.8	20.8 ± 11.4	22.9 ± 13.4*	20.8 ± 13.1	20.7 ± 11.6†
Alcohol intake,[7] servings/wk	6.3 ± 11.6	7.1 ± 13.0	6.7 ± 11.9	7.1 ± 14.1	0.4 ± 2.3	0.4 ± 2.5	0.2 ± 1.8	0.4 ± 2.5
Current smoker, % (n)	57.4 (877)	52.5 (755)*	58.9 (156)	50.7 (261)*	2.9 (44)	4.1 (75)	4.4 (9)	3.6 (26)
Activity, METs/wk	268 ± 214	228 ± 201	257 ± 225	203 ± 203*	245 ± 211	248 ± 233	207 ± 197	184 ± 206*†

[1] Values are means ± SDs unless otherwise indicated. *Different from "No infl/norm WHtR" group in pairwise testing, $P < 0.05$; †different from "No infl/high WHtR" group in pairwise testing, $P < 0.05$ (t tests for continuous variables and chi-square tests for categorical variables). CHNS, China Health and Nutrition Survey; hs-CRP, high-sensitivity C-reactive protein; ID, infectious disease; Infl, inflammation; Infl/high WHtR, hs-CRP of 3–10 mg/L and WHtR >0.5; Infl/norm WHtR, hs-CRP of 3–10 mg/L and WHtR ≤0.5; MET, metabolic equivalent task; No infl/high WHtR, WHtR >0.5 and hs-CRP <3 mg/L; No infl/norm WHtR, hs-CRP <3 mg/L and WHtR ≤0.5; norm, normal; WHtR, waist-to-height ratio.

[2] Based on tertiles of the urbanicity scale (range: 30.4–106.6), representing low (<59.0), medium (59.0–82.2), and high (>82.2) urbanicity (21).

[3] Resident of Heilongjang, Henan, Liaoning, or Shandong provinces.

[4] Derived from a scale measuring access to water and toilet facilities and exposure to excrement, livestock, particulate-generating cooking fuel, and car exhaust. Scores have a possible range of 7–22, with higher values representing better household sanitation.

[5] Occurrence of fever/sore throat/cough/asthma, diarrhea/stomachache, or other IDs in the 24 h preceding blood sample collection.

[6] More than 30% of energy from fat (23).

[7] A serving is defined as 1 bottle of beer (~10.4 g ethanol), 50 g wine (~4.8 g ethanol), or 50 g liquor (~15.8 g ethanol).

at a lower BMI than other groups (28). WHtR, therefore, may be a particularly useful measure for capturing metabolic risk among those who are younger and are not yet overweight or obese. We found that a considerable proportion of the Chinese adults in our sample had high WHtR, and this proportion increased with age from 20–25% at 18–29.9 y of age to 57–77% at ages ≥70 y. Although fewer adults had moderate inflammation, this proportion also increased with age to nearly one-third at ages ≥70 y. Despite this marked age pattern and the significantly older ages of all the risk groups compared with the healthy referent group, no significant age differences existed between men or women in the high WHtR with inflammation group and those in the high WHtR without inflammation group. The lack of significant age differences between these groups suggests that they may represent distinct risk groups, similar to those described for metabolically healthy and metabolically abnormal obesity (10, 11), rather than a single group of individuals with high WHtR who develop inflammation as they age.

The risk of high WHtR and/or inflammation varied by region and urbanicity, further suggesting that these groups differ in their exposures to urbanization and socioeconomic changes. The impacts of these geographic and socioeconomic factors differed between men and women. High WHtR, both with and without inflammation, was more likely to be seen among more affluent men living in more urban areas. Conversely, women with high WHtR without inflammation were less likely than those in the healthy referent group to live in highly urban areas or in the south, whereas women with both high WHtR and inflammation were more likely to live in moderately urban areas. The opposite associations between urbanicity and central obesity and inflammation that we found in men and women, with more urban men and fewer urban women at greater risk of high WHtR and inflammation, are similar to those previously shown for a number of other cardiometabolic risk factors (29). Previous research documented that urban Chinese men are more likely to suffer from components of the metabolic syndrome than are urban Chinese women, whereas rural Chinese women are at greater risk than rural men (29–31). These differing patterns have been proposed to reflect underlying regional and sex-based differences in exposure to "Western" diets and lifestyles (32).

Among the potential behavioral factors underlying these patterns, we found significant differences in diet and physical activity between the groups in both men and women. We tested 2 aspects of diet, fat and fiber intake, that serve as markers of a Western diet pattern (33, 34) and that have previously been shown to have significant and opposite inflammatory effects (35, 36). Contrary to our expectations, we found no independent effect of high fat consumption on the likelihood of having high WHtR and/or inflammation. Although overall fat intake has previously been used as a marker of a Western diet (33, 34) and in this sample was significantly associated with the consumption of Western-diet food items, including high-fat red meat, deep-fried wheat, and fast foods (37), the lack of expected associations between fat intake and high WHtR may indicate that this variable is too crude and that future analysis should focus on the types of fat (saturated vs. unsaturated) or sources of fat consumed (38, 39).

Our findings that women with both high WHtR and inflammation were less likely to consume high amounts of fiber than

TABLE 2 Factors distinguishing Chinese men and women participating in the CHNS with high WHtR and/or moderate inflammation from a healthy referent group[1]

	Men						Women					
	No infl/high WHtR		Infl/norm WHtR		Infl/high WHtR		No infl/high WHtR		Infl/norm WHtR		Infl/high WHtR	
	RR (95% CI)	*P*	RR (95% CI)	*P*	RR (95% CI)	*P*	RR (95% CI)	*P*	RR (95% CI)	*P*	RR (95% CI)	*P*
Model 1: sociodemographic factors												
Urbanicity[2]												
Low	Reference	—	Reference	—	Reference	—	Reference	—	Reference	—	Reference	—
Middle	1.57 (1.30, 1.88)	<0.001	1.29 (0.94, 1.77)	0.12	1.61 (1.24, 2.09)	<0.001	1.11 (0.93, 1.34)	0.25	1.07 (0.73, 1.58)	0.71	1.30 (1.02, 1.66)	0.03
High	1.57 (1.30, 1.89)	<0.001	1.09 (0.78, 1.52)	0.62	1.63 (1.26, 2.11)	<0.001	0.71 (0.59, 0.85)	<0.001	1.07 (0.74, 1.54)	0.73	0.97 (0.76, 1.23)	0.80
Income												
Low	Reference	—	Reference	—	Reference	—	Reference	—	Reference	—	Reference	—
Medium	1.26 (1.05, 1.52)	0.01	0.97 (0.71, 1.33)	0.86	1.23 (0.94, 1.59)	0.13	1.02 (0.85, 1.22)	0.83	1.09 (0.75, 1.57)	0.66	0.94 (0.75, 1.19)	0.63
High	1.37 (1.14, 1.66)	0.01	0.73 (0.52, 1.03)	0.07	1.42 (1.09, 1.83)	0.008	1.03 (0.86, 1.23)	0.78	1.15 (0.79, 1.67)	0.46	1.04 (0.82, 1.32)	0.75
North resident[3]	0.60 (0.51, 0.70)	<0.001	0.87 (0.66, 1.15)	0.33	0.68 (0.55, 0.84)	<0.001	0.81 (0.70, 0.94)	0.007	0.96 (0.71, 1.30)	0.78	0.93 (0.77, 1.13)	0.47
Model 2: behavioral and environmental factors												
ID symptoms	1.07 (0.76, 1.53)	0.70	2.78 (1.76, 4.37)	<0.001	1.93 (1.27, 2.92)	0.002	0.95 (0.69, 1.31)	0.74	2.03 (1.22, 3.38)	0.006	1.4 (0.95, 2.06)	0.09
Sanitation												
Low	Reference	—	Reference	—	Reference	—	Reference	—	Reference	—	Reference	—
Middle	1.08 (0.90, 1.30)	0.41	0.85 (0.62, 1.17)	0.33	1.29 (0.99, 1.69)	0.06	0.9 (0.75, 1.08)	0.25	0.85 (0.58, 1.25)	0.41	0.99 (0.77, 1.26)	0.92
High	1.26 (1.00, 1.58)	0.06	0.77 (0.50, 1.17)	0.22	1.67 (1.21, 2.30)	0.002	0.66 (0.52, 0.83)	<0.001	0.8 (0.50, 1.27)	0.35	0.74 (0.55, 1.00)	0.05
High fat intake[4]	1.05 (0.89, 1.23)	0.59	1.13 (0.85, 1.50)	0.40	1.03 (0.83, 1.29)	0.77	0.98 (0.84, 1.15)	0.83	1.19 (0.86, 1.64)	0.29	1.13 (0.92, 1.39)	0.24
Fiber intake[5]												
<25th percentile	Reference	—	Reference	—	Reference	—	Reference	—	Reference	—	Reference	—
25th–75th percentile	1.03 (0.86, 1.24)	0.75	1.08 (0.79, 1.50)	0.62	0.92 (0.72, 1.18)	0.51	1.18 (0.98, 1.42)	0.09	1.05 (0.72, 1.51)	0.81	1.01 (0.80, 1.28)	0.93
>75th percentile	1.24 (1.00, 1.55)	0.05	0.85 (0.56, 1.28)	0.43	1.14 (0.84, 1.54)	0.39	1.34 (1.08, 1.66)	0.008	0.92 (0.59, 1.44)	0.71	1.04 (0.79, 1.38)	0.76
Physical activity												
<25th percentile	Reference	—	Reference	—	Reference	—	Reference	—	Reference	—	Reference	—
25th–50th percentile	0.95 (0.74, 1.21)	0.66	1 (0.64, 1.56)	1.0	0.9 (0.66, 1.23)	0.51	0.76 (0.61, 0.94)	0.01	0.81 (0.53, 1.22)	0.32	0.68 (0.53, 0.88)	0.004
>50th–75th percentile	0.94 (0.74, 1.19)	0.61	1.01 (0.66, 1.54)	0.96	0.74 (0.54, 1.01)	0.06	0.76 (0.60, 0.97)	0.03	0.78 (0.48, 1.25)	0.30	0.70 (0.52, 0.95)	0.02
>75th percentile	0.58 (0.45, 0.75)	<0.001	0.93 (0.60, 1.45)	0.76	0.53 (0.37, 0.75)	<0.001	0.95 (0.75, 1.21)	0.69	0.70 (0.42, 1.15)	0.16	0.52 (0.38, 0.72)	<0.001
Alcohol intake[6]												
None	Reference	—	Reference	—	Reference	—	Reference	—	Reference	—	Reference	—
Low	1.30 (1.08, 1.57)	0.005	0.98 (0.70, 1.37)	0.91	1.1 (0.85, 1.42)	0.48	1.08 (0.84, 1.40)	0.55	0.72 (0.39, 1.30)	0.27	0.91 (0.63, 1.29)	0.59
High	1.37 (1.14, 1.65)	0.001	1.07 (0.77, 1.49)	0.67	1.21 (0.94, 1.57)	0.14	—	—	—	—	—	—
Current smoker	0.80 (0.68, 0.93)	0.004	1.14 (0.86, 1.50)	0.37	0.82 (0.66, 1.01)	0.07	0.80 (0.54, 1.18)	0.26	1.12 (0.53, 2.37)	0.77	0.62 (0.37, 1.04)	0.07
Age group												
18–29.9 y	Reference	—	Reference	—	Reference	—	Reference	—	Reference	—	Reference	—
30–39.9 y	2.60 (1.88, 3.60)	<0.001	1.51 (0.86, 2.62)	0.15	1.97 (1.19, 3.26)	0.009	2.68 (1.91, 3.75)	<0.001	1.05 (0.59, 1.85)	0.87	2.36 (1.33, 4.20)	0.003
40–49.9 y	3.57 (2.61, 4.87)	<0.001	1.41 (0.82, 2.43)	0.22	3.56 (2.23, 5.67)	<0.001	5.51 (4.01, 7.58)	<0.001	1.52 (0.90, 2.57)	0.12	5.11 (2.99, 8.71)	<0.001

(Continued)

TABLE 2 *Continued*

	Men						Women					
	No infl/high WHtR		Infl/norm WHtR		Infl/high WHtR		No infl/high WHtR		Infl/norm WHtR		Infl/high WHtR	
	RR (95% CI)	*P*	RR (95% CI)	*P*	RR (95% CI)	*P*	RR (95% CI)	*P*	RR (95% CI)	*P*	RR (95% CI)	*P*
50–59.9 y	4.08 (3.00, 5.55)	<0.001	1.83 (1.08, 3.09)	0.02	3.86 (2.44, 6.11)	<0.001	9.38 (6.78, 12.98)	<0.001	1.88 (1.09, 3.24)	0.02	13.40 (7.92, 22.66)	<0.001
60–69.9 y	4.86 (3.51, 6.73)	<0.001	2.42 (1.40, 4.19)	0.002	4.01 (2.47, 6.50)	<0.001	13.57 (9.48, 19.44)	<0.001	3.00 (1.66, 5.43)	<0.001	23.97 (13.86, 41.5)	<0.001
≥70 y	3.82 (2.62, 5.58)	<0.001	4.28 (2.39, 7.68)	<0.001	6.01 (3.60, 10.02)	<0.001	12.47 (8.43, 18.43)	<0.001	3.08 (1.61, 5.87)	0.001	23.24 (13.13, 41.1)	<0.001

[1] Values are from a multinomial logistic regression model compared with a healthy referent group. CHNS, China Health and Nutrition Survey; hs-CRP, high-sensitivity C-reactive protein; ID, infectious disease; Infl, inflammation; Infl/high WHtR, hs-CRP of 3–10 mg/L and WHtR >0.5; Infl/norm WHtR, hs-CRP of 3–10 mg/L and WHtR ≤0.5; No infl/high WHtR, WHtR >0.5 and hs-CRP <3 mg/L; No infl/norm WHtR, hs-CRP <3 mg/L and WHtR ≤0.5; norm, normal; WHtR, waist-to-height ratio.
[2] Based on tertiles of the urbanicity scale (range: 30.4–106.6), representing low (<59.0), medium (59.0–82.2), and high (>82.2) urbanicity (21).
[3] Resident of Heilongjang, Henan, Liaoning, or Shandong provinces.
[4] More than 30% of energy from fat (23).
[5] Categories derived from a sample distribution of fiber in grams per 100 kcal.
[6] Categories indicate no consumption of alcohol per week; low, ≤7 servings/wk for men or any servings/wk for women; high, >7 servings/wk for men. A serving was defined as 1 bottle of beer (~10.4 g ethanol), 50 g wine (~4.8 g ethanol), or 50 g liquor (~15.8 g ethanol).

women with high WHtR without inflammation align with previous cross-sectional and longitudinal studies documenting an inverse association between fiber intake and CRP concentrations (40, 41). Although the finding that those with high WHtR consume more fiber per 100 kcal than healthy controls seems counterintuitive, these results may reflect the direct and indirect roles of fiber as both a nutrient and a marker of overall diet. Dietary fiber may exert anti-inflammatory effects directly by reducing insulin production (40), lowering cholesterol (40), or altering the gut microbiome and its metabolites (42). Indirectly, low-fiber diets have been associated with a greater intake of refined carbohydrates, hyperglycemia, and the production of proinflammatory cytokines in the United States and other Western countries (40). Higher fiber intake in China, however, has been associated with higher energy and fat intake (43) and increased intakes of vegetables, fruits, and fast foods (44), indicating that the relation between fiber intake and dietary patterns varies by context. Further research is needed to identify the dietary patterns associated with fiber intake in Chinese adults and the consequences of these differing dietary patterns for central obesity and inflammation.

Together with fiber intake, physical activity level was an important factor distinguishing the high WHtR groups from the healthy referent group. Not surprisingly, men and women with high WHtR were less physically active. More importantly, however, we found that women high WHtR and inflammation were less likely to perform higher levels of physical activity than women with high WHtR without inflammation, supporting previous research that physical fitness distinguishes metabolically healthy obese and metabolically abnormal individuals (40, 45, 46) and that increasing physical activity may be an important strategy for lowering CRP concentrations (47).

In addition to these obesogenic exposures, markers of pathogen exposure also differentiated the groups. Infectious illness symptoms were the main risk factor distinguishing those with inflammation and normal WHtR from the healthy referent group. Concerns have been raised that individuals exposed to both obesogenic and pathogenic environments may be at elevated risk of the development of inflammation and its attendant cardiovascular consequences, because both types of exposures upregulate inflammatory pathways (14, 15). Our results indeed suggest that those at the greatest risk (high WHtR with inflammation) are more likely to have both pathogenic and obesogenic exposures. Among men, ID symptoms were the main characteristic distinguishing those with high WHtR and inflammation from those with high WHtR without inflammation. Among women, those with high WHtR and inflammation were more likely to have infectious illness symptoms and less likely to have higher amounts of fiber intake and physical activity. Taken together, these results suggest that the dual exposure to obesogenic lifestyles (poorer fiber intake and lower physical activity) and pathogenic exposures (poorer household sanitation and more frequent infectious illness) may place individuals at risk of the development of central obesity and metabolic abnormalities.

The CHNS provides high-quality measures of dietary, behavioral, and environmental exposures collected across a wide range of ages and socioeconomic and geographic contexts, permitting us to examine a number of potential risk factors for high WHtR and inflammation. Height and waist circumference were measured by trained study personnel, and detailed diet measures were collected within the household, limiting the potential for recall bias. Individual diets were measured by using 3 d of 24-h recalls and adjusted for expected individual consumption from the weighed household inventory if there was a large difference in

TABLE 3 Factors distinguishing Chinese men and women participating in the CHNS with high WHtR and moderate inflammation from those with high WHtR without moderate inflammation[1]

	Men with infl/high WHtR		Women with infl/high WHtR	
	OR (95% CI)	*P*	OR (95% CI)	*P*
Model 1: sociodemographic factors				
Urbanicity[2]				
Low	Reference	—	Reference	—
Middle	1.01 (0.78, 1.31)	0.93	1.15 (0.92, 1.43)	0.23
High	1.02 (0.79, 1.33)	0.87	1.33 (1.06, 1.67)	0.01
Income				
Low	Reference	—	Reference	—
Medium	0.96 (0.74, 1.25)	0.79	0.93 (0.75, 1.15)	0.49
High	1.03 (0.79, 1.33)	0.85	1.03 (0.82, 1.28)	0.82
North resident[3]	1.15 (0.93, 1.41)	0.20	1.14 (0.95, 1.36)	0.17
Model 2: behavioral and environmental factors				
ID symptoms	1.73 (1.15, 2.61)	0.009	1.45 (1.01, 2.07)	0.04
Sanitation				
Low	Reference	—	Reference	—
Middle	1.17 (0.89, 1.54)	0.25	1.11 (0.89, 1.39)	0.37
High	1.28 (0.93, 1.76)	0.13	1.10 (0.83, 1.45)	0.52
High fat intake[4]	0.99 (0.80, 1.23)	0.94	1.15 (0.95, 1.39)	0.15
Fiber intake[5]				
<25th percentile	Reference	—	Reference	—
25th–75th percentile	0.88 (0.69, 1.14)	0.34	0.86 (0.69, 1.08)	0.20
>75th percentile	0.92 (0.69, 1.24)	0.61	0.77 (0.60, 1.00)	0.049
Physical activity				
<25th percentile	Reference	—	Reference	—
25th–50th percentile	0.96 (0.70, 1.32)	0.83	0.90 (0.71, 1.14)	0.40
>50th–75th percentile	0.80 (0.58, 1.09)	0.16	0.93 (0.70, 1.24)	0.61
>75th percentile	0.91 (0.64, 1.30)	0.61	0.55 (0.41, 0.73)	<0.001
Alcohol intake[6]				
None	Reference	—	Reference	—
Low	0.84 (0.65, 1.09)	0.19	0.84 (0.60, 1.18)	0.32
High	0.88 (0.68, 1.14)	0.35	—	—
Current smoker	1.03 (0.83, 1.27)	0.80	0.75 (0.47, 1.21)	0.24
Age group				
18–29.9 y	Reference	—	Reference	—
30–39.9 y	0.74 (0.43, 1.27)	0.27	0.88 (0.47, 1.65)	0.68
40–49.9 y	0.97 (0.59, 1.60)	0.90	0.89 (0.50, 1.60)	0.70
50–59.9 y	0.93 (0.57, 1.53)	0.77	1.37 (0.77, 2.43)	0.28
60–69.9 y	0.81 (0.48, 1.36)	0.43	1.69 (0.95, 3.02)	0.08
≥70 y	1.52 (0.87, 2.63)	0.14	1.80 (0.99, 3.26)	0.05

[1] Values are from logistic regression models controlling for age group compared with the high WHtR without inflammation (WHtR >0.5 and hs-CRP <3 mg/L) referent group. CHNS, China Health and Nutrition Survey; hs-CRP, high-sensitivity C-reactive protein; ID, infectious disease; Infl, inflammation; Infl/high WHtR, hs-CRP of 3–10 mg/L and WHtR >0.5; WHtR, waist-to-height ratio.

[2] Based on tertiles of the urbanicity scale (range: 30.4–106.6), representing low (<59.0), medium (59.0–82.2), and high (>82.2) urbanicity (21).

[3] Resident of Heilongjang, Henan, Liaoning, or Shandong provinces.

[4] More than 30% of energy from fat (23).

[5] Categories derived from a sample distribution of fiber in grams per 100 kcal.

[6] Categories indicate no consumption of alcohol per week; low, ≤7 servings/wk for men or any servings/wk for women; high, >7 servings/wk for men. A serving was defined as 1 bottle of beer (~10.4 g ethanol), 50 g wine (~4.8 g ethanol), or 50 g liquor (~15.8 g ethanol).

consumption between the household and individual levels (24, 48, 49). Along with these strengths, our study has several limitations. Most important, hs-CRP was only measured at 1 point in time, limiting our ability to distinguish between chronic and acute inflammation. We are unable to definitively state whether elevated hs-CRP concentrations represent chronically elevated baseline inflammation or recovery from a recent illness. To mitigate the influence of recent illness, we limited the sample to those with hs-CRP concentrations <10 mg/L, a commonly used cutoff for acute infection (19). We also used participant infectious illness symptoms (cough, diarrhea, etc.) in the 24 h preceding blood collection as a marker of pathogen burden. This measure of infectious illness is a crude one and cannot distinguish between acute and chronic exposure; however, these symptoms were not associated with hs-CRP >10 mg/L and may better reflect longer-term exposure. We chose to model the effects of distal (i.e., socioeconomic, geographic, etc.) and proximal (i.e., sanitation, diet, and physical activity) risk factors separately because we hypothesized that the effects of the distal factors would be mediated by the more proximal factors in the pathway to inflammation and high

WHtR. Although it is possible that some of the effects of the proximal factors could be due to not controlling for the socio-demographic and geographic measures, our sensitivity analysis yielded few differences between the separate and full models.

In summary, we documented that the prevalence of high WHtR and moderate inflammation increases with age in Chinese men and women. Pathogen burden, measured through the presence of ID symptoms, was the main risk factor distinguishing those with inflammation without high WHtR from the healthy referent group. Both pathogenic and obesogenic risk factors distinguished those with central obesity with and without inflammation from the healthy referent group. Compared with those with high WHtR without inflammation, women with high WHtR and inflammation had greater ID symptomology, lower fiber intake, and lower levels of physical activity. These results highlight the importance of examining context-specific risk factors for central obesity and inflammation and suggest that both pathogenic and obesogenic environmental risk factors should be targeted for the prevention of the development of cardiometabolic disease.

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